## *In the Specification*

On page 4, amend the paragraph at lines 10-11 to add a period at the end of the sentence as follows:

Fig. 1 Scheme of the method for detection of thrombolytic action of drugs *in vivo* in rats (according to Gryglewski).

On page 4, amend the paragraph at lines 12-13 to add a period at the end of the sentence as follows:

Fig. 2 Thrombolytic response induced by intravenous administration of MNA+ in vivo (30 mg/kg).

On page 4, amend the paragraph at lines 14-15 to add a period at the end of the sentence as follows:

Fig. 3 Changes in plasma levels of 6-keto-$PGF_{1\alpha}$ (●) and $TXB_2$ (○) after intravenous administration of MNA+ (30 mg/kg).

On page 4, amend the paragraph at lines 20-21 to add a period at the end of the sentence as follows:

Fig. 6 Thrombolytic response induced by intravenous administration of $MAP^+$ in vivo (30 mg/kg).

On page 4, amend the paragraph at lines 22-23 to add a period at the end of the sentence as follows:

Fig 7 Changes in plasma levels of 6-keto-$PGF_{1\alpha}$ (●) and $TXB_2$ (○) after intravenous administration of $MAP^+$ in vivo (30 mg/kg).

On page 4, amend the paragraph at lines 24-25 to add a period at the end of the sentence as follows:

Fig. 8 Thrombolytic response induced by intravenous administration of $MNAF^+$ (30 mg/kg).

On page 4, amend the paragraph at lines 26-27 to add a period at the end of the sentence as follows:

Fig. 9 Lack of effect of $MNA^+$ on collagen-induced aggregation of platelets (1 mg/ml).

On page 4, amend the paragraph that beings on line 28 to add a period at the end of the sentence as follows:

Fig. 10 Lack of effect of $MNA^+$ on latex-induced activation of neutrophils.

At page 7, amend the last paragraph as below:

The invention in the second aspect provides a method of treatment and/or prevention of conditions or diseases associated with dysfunction of vascular endothelium, oxidative stress, and/or insufficient production of endothelial $PGI_2$ (associated or not with hypercholesterolemia, hypertriglyceridemia or a low HDL level), in particular such as discussed above, comprising administration to a subject in [[a]] need of such treatment a therapeutically effective amount of a quaternary pyridinium salt of formula I as defined above.

On page 12, amend the third full paragraph as follows:

During the initial 20-30 min of superfusion the strip gained ~~was gaining in weight by~~ 80-120 mg in weight because of the deposition of platelet-rich thrombi [[_]] and then in control conditions stayed unchanged during the next 3-5 hrs. A thrombolytic ~~Thrombolytic~~ response was detected by a fall in weight ~~weigth~~ of a thrombi. Arterial blood pressure was also monitored, so this model enabled the analysis of thrombolytic and hypotensive action of a compound (Fig. 1).

At page 12, amend the last full paragraph as follows:

The analysis of the thrombolytic response in this experimental set-up was complemented by the assay of 6-keto-$PGF_{1\alpha}$, $TXB_2$ and $PGE_2$ in arterial blood plasma. For this purpose blood samples (500 µl) were collected in EPPENDORF® [[Eppendorff]] tubes with indomethacin to yield its final concentration of 10 µM, and EDTA to yield the final concentration of 1 mM. Then, the blood samples were spun ~~spinned~~ for 5 min at 2.000 x g. Plasma samples were stored at -70°C. The prostanoids were assayed using the enzyme immunoassay kits (Cayman Chemical Co, Ann Arbor, MI).

At page 12, amend the paragraph that being on line 30 as follows:

Intravenous administration of MNA (3-30 mg/kg) produced a concentration-dependent thrombolysis in Wistar rats with extracorporal circulation. A maximum response was observed at the $MNA^+$ dose of 30 mg/kg. Single injection of $MNA^+$ at a dose of 30 mg/kg induced a long-lasting thrombolytic response at the level of 42± 4 % and remained at approximately the same level for 2-3 hours of the observation period. In contrast to $MNA^+$, nicotinamide, nicotinic acid, trigonelline and 2-PYR (endogenous metabolite of $MNA^+$), each of them at 30 mg/kg, failed to induce a significant thrombolytic response. Nicotinamide and nicotinic acid-induced responses were transient (less then 15-20 minutes) and at their maximum amounted merely to 9±0.6 %, 5±0.9 %, respectively). Trigonelline did not produce any thrombolytic response and response to 2-PYR was also very weak (<10 %) and transient (<15 min). The potency and duration of

thrombolytic responses to $MNA^+$, nicotinamide and nicotinic acid correlated with a pattern of 6-keto-$PGF_{1\alpha}$ release to arterial plasma induced by these compounds. $MNA^+$ (30 mg/kg) induced a substantial increase in levels of 6-keto-$PGF_{1\alpha}$ as early as 15 minutes after drug injection (from 104±7 to 460±58 pg/ml) which then [[than]] reached its plateau of around 400 pg/ml for at least one hour. On the other hand neither $TXB_2$ nor $PGE_2$ levels changed significantly in response to $MNA^+$. The sluggish ~~Sluggish~~ rise in $TXB_2$ levels was time-dependent and observed also after saline injection. Levels of 6-keto-$PGF_{1\alpha}$ did not increase after injection of nicotinamide or after injection of nicotinic acid (30 mg/kg).

At page 14, line 10, amend the title of Example 2 as follows:

An anti-arthrogenic ~~ant-arthrogenic~~ effect in vivo in patients